Blood Lead Levels in the US Population

Phase 1 of the Third National Health and Nutrition Examination Survey (NHANES III, 1988 to 1991)

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Objective.—To determine mean blood lead levels and their sociodemographic correlates in the US population.

Design.—Nationally representative cross-sectional health examination survey that included measurements of venous blood lead.

Participants.—A total of 13 201 persons aged 1 year and older examined during phase 1 of the third National Health and Nutrition Examination Survey (1988 to 1991).

Results.—The overall mean blood lead level for the US population was 0.14 μmol/L (2.8 μg/dL). Blood lead levels were consistently higher for younger children than for older children, for older adults than for younger adults, for males than for females, for blacks than for whites, and for central-city residents than for non–central-city residents. Other correlates of higher blood lead levels included low income, low educational attainment, and residence in the Northeast region of the United States. National estimates for children 1 to 5 years of age indicate that 8.9%, or approximately 1.7 million children, have blood lead levels 0.48 μmol/L (10 μg/dL) or greater. These levels are high enough to be of health concern under 1991 Centers for Disease Control and Prevention guidelines.

Conclusions.—The low overall mean blood lead levels demonstrate a major public health success in primary prevention efforts. However, exposure to lead at levels that may adversely affect the health of children remains a problem especially for those who are minority, urban, and from low-income families. Strategies to identify the most vulnerable risk groups are necessary to further reduce lead exposure in the United States.

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THE PERSISTENCE of lead in the environment poses an ongoing challenge to the field of public health. A toxicant whose deleterious health effects have been known since antiquity, lead continues to attract national attention. The pervasiveness of lead is illustrated by reports of sources of exposure that range from paint removed during the renovation of a Victorian farmhouse[1] to contaminated soil concentrated in urban play areas[2] to traditional medicine ingested for a stomach ailment.[3]

Strategies to eliminate lead poisoning include reducing sources of exposure, increasing safe and effective abatement programs, and identifying persons at risk.[4] Surveillance plays an important role in documenting lead exposure by characterizing vulnerable population groups and assessing the effectiveness of intervention efforts. Blood lead levels measured as part of the National Health and Nutrition Examination Surveys (NHANES) conducted by the National Center for Health Statistics/Centers for Disease Control and Prevention (NCHS/CDC) have contributed to the national surveillance of lead exposure in the United States. The NHANES provide blood lead level estimates for population subgroups by age, sex, race/ethnicity, income level, urban status, and region of the country. The second National Health and Nutrition Examination Survey (NHANES II, 1976 to 1980) yielded the first national estimates of blood lead levels.[5] Estimates were also produced from the Hispanic Health and Nutrition Examination Survey (1982 through 1984), a special survey of Mexican Americans, Cubans, and Puerto Ricans.[6]

See also pp 284 and 315.

This article presents blood lead levels from phase 1 of the third National Health and Nutrition Examination Survey (NHANES III phase 1, 1988 to 1991), the most recent of the NHANES. The distribution of blood lead levels is described by sociodemographic characteristics for persons aged 1 year and older. A new feature of the survey was the extension of sampling to persons older than 74 years, which enabled the determination of blood lead levels in the growing subgroup of older Americans.

METHODS AND PROCEDURES

NHANES III Sample Design

The NHANES III, a 6-year survey measuring the health and nutritional status of the civilian noninstitutionalized US population aged 2 months and older, is being conducted by the NCHS/CDC from 1988 to 1994. National population estimates as well as estimates for the three largest race/ethnicity subgroups in the US population (non-Hispanic white, non-Hispanic black, and Mexican American) can be derived from each of two individual 3-year phases or from the full 6-year survey. Phase 1 was conducted from October 1988 through October 1991.

The sampling scheme for NHANES III was based on a complex multistage area probability design. Children younger than 5 years, adults aged 60 years and older, blacks, and Mexican Americans were

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oversampled. A detailed description of the sample design has been published.[7]

Data were collected through a household interview and a standardized physical examination conducted in a mobile examination center. Sociodemographic information and medical histories of the survey participant and the family were collected during the household interview.

Laboratory Methods

During the physical examination, a 1-mL sample of ethylenediaminetetraacetic acid–anticoagulated whole blood was obtained by venipuncture from participants aged 1 year and older. Blood specimens were frozen and shipped on dry ice to the NHANES laboratory, Division of Environmental Health Laboratory Sciences, National Center for Environmental Health, CDC, Atlanta, Ga, for analysis. Specimens remained frozen at −20°C until analysis.

Lead was measured by graphite furnace atomic absorption spectrophotometry (GFAAS) using the method of Miller et al.[8] The lead content was determined using GFAAS with deuterium background correction (Perkin-Elmer Model 5000). This method has been optimized for sensitivity at lower blood lead levels resulting in a detection limit of 0.05 μmol/L (1.0 μg/dL). In all statistical analyses, blood lead levels less than 0.05 μmol/L (1.0 μg/dL) (6.5% of the samples) were assigned a level of 0.02 μmol/L (0.5 μg/dL).

Analysis of each specimen was performed in duplicate, and the mean of the duplicate measurements was reported. All specimens containing lead concentrations greater than 0.72 μmol/L (15 μg/dL) or less than 0.07 μmol/L (1.4 μg/dL) were rediluted and reanalyzed by GFAAS for confirmation. A comparison between GFAAS and inductively coupled mass spectrometry showed good agreement at very low concentrations of lead.

Bench and blind quality control (QC) procedures were used to assure quality of the lead analyses.[9] Four bench QC samples were inserted in each run of 60 specimens to evaluate method performance on the day of analysis. In addition, 5% of the samples were blind QC samples, which appeared as a regular unknown sample to the analyst. Blind QC results were monitored by a scientist not involved in the analysis of samples. The QC results showed no statistically significant trends in blood lead level measurement during the 3-year study period of October 1988 through October 1991.

Demographic and Socioeconomic Covariates

Age was reported at the time of the household interview as the age in years at last birthday. Age categories used in analyses were 1 to 2 years, 3 to 5 years, 6 to 11 years, 12 to 19 years, 20 to 49 years, 50 to 69 years, and 70 years and older. In the regression analyses, the first two age categories (1 to 5 years) and the last two age categories (≥50 years) were collapsed.

A composite race/ethnicity variable, based on reported race and ethnicity, was created to define three major race/ethnicity groups: non-Hispanic black, non-Hispanic white, and Mexican American. Persons from other race/ethnicity groups were included in the overall blood lead estimates but not in the estimates stratified by race/ethnicity due to limited sample size.

Education was dichotomized as high school graduate or less than a high school graduate. For adults aged 20 years and older, the variable reflected the education of the examinee. For children and youths aged 1 to 19 years, the education of the adult reference person was used in the analyses. The adult reference person was defined as one of the persons in the household who owns or rents the home.

Income level was defined by the poverty-income ratio (PIR): the total family income divided by the poverty threshold for the year of the interview. Income included the total family wages, salaries, Social Security and retirement benefits, and any other earnings received during the 12 months prior to the interview. The poverty threshold, determined annually by the US Bureau of the Census,[10-12] is adjusted for family size. The PIR was used both as a continuous variable and a categorical variable, defined as low ($0<PIR<1.30$), mid ($1.30\leq PIR<3.00$), and high ($PIR\geq 3.00$). These categories were selected in part to be consistent with major government food assistance programs that use a PIR of 1.30 to determine eligibility.[13]

Urban status was defined by population size and place of residence. Population was dichotomized as 1 million or more or less than 1 million. The place of the residence was designated as within or not within the central city of a standard metropolitan area. Population and place of residence were combined to create a single urban indicator with three levels: population of 1 million or more and central city; population of less than 1 million and central city; and non–central city.

Region, defined by the US Bureau of the Census, describes the geographic area of the United States where the examinee resided based on the categorization of states as Northeast, Midwest, South, and West.

Response Rates and Potential Nonresponse Bias

The current analysis was based on data from examinees aged 1 year and older. Of the 19 103 persons aged 1 year and older selected for the survey, 16 341 (86%) were interviewed and eligible for an examination. Of those eligible, 14 870 (91%) were examined. Blood lead determinations were available for 13 201 of the eligible examinees, representing 89% of the persons examined and 69% of the persons selected for the survey. Young children and older adults were more likely to have missing lead values.

To determine the potential effects of differential nonresponse on mean blood lead levels, an analysis of persons with lead values and persons without lead values (but interviewed) was conducted with respect to major demographic characteristics (age, sex, race/ethnicity, education, region, PIR, household size, location of residence, urban status, and sex and marital status of the adult reference person) as well as other health risk factors (overall health status, age of house, season, ever tested for lead, and identified or treated for lead poisoning). For each variable, the observed mean blood lead level in the examined sample was compared with the expected mean blood lead level in the interviewed sample, after adjusting for that variable. Using a method described by Flegal et al,[14] it was assumed that no significant differential in mean blood lead level resulting from nonresponse was present if the observed estimate was within 10% of the expected estimate. An analysis was also conducted to examine the potential bias of prevalence of high blood lead levels as defined by two values: 0.48 μmol/L (10 μg/dL) and 0.72 μmol/L (15 μg/dL). No bias in the mean blood lead levels or in the prevalence of high blood lead levels due to nonresponse could be detected.

Within-Person Variation

The effect of within-person variation on the prevalence of elevated blood lead level (≥0.48 μmol/L [≥10 μg/dL]) was determined to further assess the reliability of the estimates. Large within-person variation can distort prevalence estimates by increasing the total variance of the distribution.[15] A sample of examination participants aged 6 years and older (n=1149) provided blood specimens on two separate occasions in NHANES III phase 1 that were analyzed for blood lead level. Following a method described by Sempos et al,[15] an adjusted prevalence estimate was calculated to evaluate the potential effect of within-person variation. The correlation between the two blood lead values was 0.94. Because the adjusted prevalence of elevated blood lead level (4.2%) differed little from the unadjusted prevalence (4.4%), it was not considered necessary to adjust for within-person variation.

Statistical Analysis

Statistical analyses were conducted using SAS.[16] Survey sample weights were used for all analyses to produce estimates that were representative of the noninstitutionalized civilian US population. SUDAAN,[17] a statistical software package that incorporates the sample weights and adjusts for the complex sample design of the survey, was used to calculate appropriate SEs.

Geometric mean blood lead levels were calculated by taking the antilog of the mean of $\log_{10}$ of the measured lead values. In this article, elevated blood lead level was defined as 0.48 μmol/L (10 μg/dL) or greater for persons of all ages. The definition was selected to be consistent with the lowest blood lead intervention level designated in the current CDC guidelines for preventing lead poisoning in young children.[18]

Multivariate linear regression analyses were performed to determine the relation between blood lead level and sociodemographic variables. Separate models were run for five age categories: 1 to 5 years, 6 to 11 years, 12 to 19 years, 20 to 49 years, and 50 years and older. Log_{10} lead was used as the dependent variable. Independent variables included sex, age (continuous), race/ethnicity, PIR, education, urban status, and region. Marital status and sex of the adult reference person, household size, age of house, and a number of interaction terms were also examined but not included in the final models because they did not significantly add to the fit of the models. Persons who had missing values for education (1.1%), PIR (10.5%), or urban status (2.4%) were not included in the regression models.

RESULTS

Mean Blood Lead Levels by Age

Geometric means and 95% confidence intervals of blood lead levels are presented by age category along with population estimates in Table 1. Among children and youths, the geometric mean was highest for 1- to 2-year-olds and lowest for youths aged 12 to 19 years. Among adults, mean blood lead levels were highest in the older age groups. The mean blood lead level of the oldest adults was almost as high as that of the youngest children.

Mean Blood Lead Levels by Sex, Age, and Race/Ethnicity

Geometric mean blood lead levels varied by sex, age, and race/ethnicity as shown in Figs 1 and 2. Variations by age were similar for males and females; however, males showed consistently higher mean blood lead levels than did females except at the youngest ages (1 to 2 years), in whom blood lead levels were similar (Fig 1). Beginning at approximately 12 years of age, sex differences in blood lead levels were pronounced, with mean blood lead levels of males being greater than levels of females by 0.05 to 0.10 μmol/L (1 to 2 μg/dL). Mean blood lead levels among non-Hispanic blacks were consistently higher than those of non-Hispanic whites, although the pattern of variability was similar for both groups (Fig 2). Mean blood lead levels of Mexican Americans were slightly higher than those of non-Hispanic whites until age 60 years. The largest differences between the three race/ethnicity groups occurred at younger than 10 years; blood lead levels of non-Hispanic black children were 0.07 to 0.10 μmol/L (1.5 to 2.0 μg/dL) higher than the blood lead levels of Mexican-American children and at least 0.10 μmol/L (2 μg/dL) higher than those of non-Hispanic white children. Blood lead levels of non-Hispanic blacks older than 50 years consistently exceeded the levels of non-Hispanic whites and Mexican Americans.

The difference in mean blood lead levels by race/ethnicity persisted when stratified by sex, particularly for males (Table 2). For females, differences in mean blood lead levels by race/ethnicity were comparable with those of males but not as pronounced. Among older males, the mean blood lead levels of Mexican Americans were similar to levels of non-Hispanic whites and were on the average 0.10 μmol/L (2 μg/dL) lower than levels of non-Hispanic blacks.

Table 1.—Weighted Geometric Means and 95% Confidence Intervals (CIs) of Blood Lead Levels for Persons Aged 1 Year and Older by Age Category: United States, 1988 to 1991

Age, y	No. Examined	Population Estimate, Thousands*	Geometric Mean, μmol/L (μg/dL)	95% CI, μmol/L (μg/dL)
1-2	925	7476	0.19 (4.1)	0.18-0.22 (3.7-4.5)
3-5	1309	11 165	0.17 (3.4)	0.15-0.19 (3.0-3.8)
6-11	1587	21 748	0.12 (2.5)	0.11-0.13 (2.2-2.7)
12-19	1376	27 293	0.08 (1.6)	0.07-0.09 (1.4-1.9)
20-49	4320	112 283	0.13 (2.6)	0.12-0.14 (2.5-2.8)
50-69	2071	42 802	0.19 (4.0)	0.18-0.20 (3.8-4.2)
≥70	1613	19 440	0.19 (4.0)	0.18-0.21 (3.7-4.3)
All	**13 201**	**242 207**	0.14 (2.8)	0.13-0.15 (2.7-3.0)

*US Bureau of the Census, *Current Population Survey*, 1990.

Prevalence of Elevated Blood Lead Levels (≥0.48 μmol/L [≥10 μg/dL])

The overall prevalence of elevated blood lead levels (≥0.48 μmol/L [≥10 μg/dL]) was 4.5% (Table 3). Children aged 1 to 2 years had the highest prevalence, and youths aged 12 to 19 years had the lowest prevalence. Among adults, those aged 20 to 49 years had a prevalence only half that of adults in the older age groups.

The proportion of children aged 1 to 5 years with elevated blood lead levels varied by race/ethnicity (Table 4). The prevalence of elevated blood lead levels among 1- to 2-year-old non-Hispanic black children (21.6%; SE, 3.1%) was 2.5 times higher than the prevalence among non-Hispanic white children (8.5%; SE, 1.7%) and twice as high as among Mexican-American children (10.1%; SE, 1.9%). The prevalence among non-Hispanic black children aged 3 to 5 years (20.0%; SE, 3.1%) was similar to that of younger non-Hispanic black children but considerably higher than the prevalence among non-Hispanic white children (3.7%; SE, 1.8%) and Mexican-American children (6.8%; SE, 1.4%) in the same age category.

The prevalence of elevated blood lead levels among children aged 1 to 5 years increased with decreasing family income (Table 5). The prevalence for children from low-income families (16.3%) was four times higher than the prevalence for children from high-income families (4.0%). Non-Hispanic black children from low-income families had the highest proportion of elevated blood lead levels (28.4%). Among children from mid- and high-income families, the variability in the prevalence of elevated blood lead levels by race/ethnicity was less pronounced.

The prevalence of elevated blood lead levels was higher for children living in more urbanized areas (Table 5). By race/ethnicity, non-Hispanic black children residing in central cities with populations 1 million or greater had the highest prevalence of elevated levels (36.7%), more than seven times the prevalence for non-Hispanic white children residing in noncentral cities. A high proportion of Mexican-American children residing in the most urbanized areas also had elevated blood lead levels (17.0%). It should be noted that comparisons of estimates across race/ethnicity were limited due to the small sample of non-Hispanic white children living in the most urbanized areas.

Among adults (aged 20 years and older) the variability in the prevalence of elevated blood lead levels by race/ethnicity was similar to that of children. The prevalence was higher for non-His-

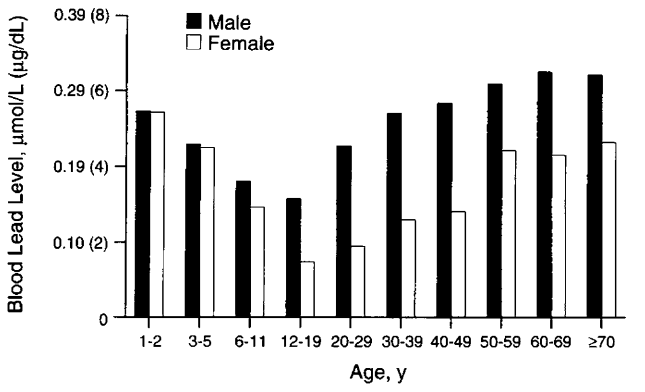

Fig 1.—Weighted geometric mean blood lead levels for persons aged 1 year and older by age and sex: United States, 1988 to 1991.

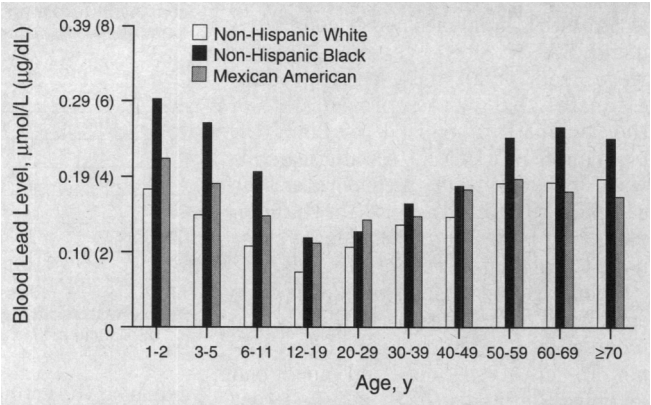

Fig 2.—Weighted geometric mean blood lead levels for persons aged 1 year and older by age and race/ethnicity: United States, 1988 to 1991.

Table 2.—Weighted Geometric Means and 95% Confidence Intervals (CIs) of Blood Lead Levels for Persons Aged 1 Year and Older by Age Category, Sex, and Race/Ethnicity: United States, 1988 to 1991*

	Non-Hispanic White			Non-Hispanic Black			Mexican American		
Age, y	No.	Geometric Mean	95% CI	No.	Geometric Mean	95% CI	No.	Geometric Mean	95% CI
					Males				
1-2	156	0.17 (3.5)	0.15-0.20 (3.1-4.1)	137	0.30 (6.3)	0.27-0.34 (5.6-7.2)	141	0.20 (4.2)	0.16-0.26 (3.3-5.3)
3-5	182	0.14 (2.9)	0.13-0.16 (2.6-3.2)	185	0.28 (5.9)	0.24-0.33 (5.1-6.8)	232	0.19 (4.0)	0.15-0.25 (3.1-5.1)
6-11	236	0.11 (2.4)	0.10-0.13 (2.1-2.7)	208	0.21 (4.5)	0.19-0.24 (3.9-5.1)	323	0.15 (3.1)	0.12-0.19 (2.4-3.9)
12-19	201	0.10 (2.1)	0.09-0.12 (1.8-2.5)	174	0.16 (3.2)	0.14-0.18 (2.9-3.7)	254	0.16 (3.3)	0.12-0.22 (2.5-4.4)
20-49	723	0.18 (3.8)	0.17-0.19 (3.6-4.1)	606	0.21 (4.5)	0.20-0.23 (4.2-4.8)	743	0.21 (4.4)	0.18-0.24 (3.8-5.0)
50-69	520	0.22 (4.7)	0.21-0.24 (4.5-4.9)	241	0.32 (6.6)	0.28-0.36 (5.8-7.5)	265	0.22 (4.5)	0.18-0.27 (3.7-5.4)
≥70	605	0.23 (4.8)	0.22-0.24 (4.5-5.1)	111	0.33 (6.8)	0.29-0.37 (6.0-7.6)	97	0.23 (4.8)	0.20-0.26 (4.3-5.4)
All	**2623**	0.17 (3.6)	0.16-0.18 (3.4-3.8)	**1662**	0.23 (4.7)	0.21-0.25 (4.4-5.0)	**2055**	0.19 (4.0)	0.16-0.23 (3.3-4.8)
					Females				
1-2	150	0.18 (3.6)	0.15-0.21 (3.0-4.3)	144	0.28 (5.8)	0.25-0.32 (5.1-6.5)	157	0.23 (4.8)	0.21-0.25 (4.4-5.3)
3-5	170	0.14 (3.0)	0.13-0.17 (2.6-3.5)	213	0.24 (5.0)	0.22-0.27 (4.5-5.6)	275	0.17 (3.6)	0.14-0.21 (3.0-4.5)
6-11	224	0.09 (1.9)	0.08-0.11 (1.6-2.2)	182	0.18 (3.8)	0.16-0.21 (3.3-4.4)	357	0.13 (2.8)	0.12-0.16 (2.4-3.3)
12-19	237	0.05 (1.0)	0.04-0.06 (0.8-1.1)	197	0.09 (1.8)	0.08-0.10 (1.6-2.0)	254	0.07 (1.5)	0.05-0.09 (1.1-2.0)
20-49	728	0.08 (1.7)	0.08-0.09 (1.6-1.9)	623	0.11 (2.2)	0.10-0.12 (2.0-2.5)	732	0.10 (2.0)	0.08-0.12 (1.7-2.5)
50-69	477	0.15 (3.2)	0.14-0.17 (3.0-3.5)	257	0.20 (4.3)	0.19-0.22 (3.9-4.7)	255	0.16 (3.2)	0.13-0.19 (2.7-3.9)
≥70	563	0.17 (3.5)	0.15-0.18 (3.2-3.8)	136	0.20 (4.2)	0.18-0.23 (3.7-4.7)	75	0.13 (2.7)	0.10-0.16 (2.1-3.4)
All	**2549**	0.10 (2.1)	0.09-0.11 (1.9-2.2)	**1752**	0.13 (2.8)	0.13-0.14 (2.6-3.0)	**2105**	0.11 (2.3)	0.09-0.13 (1.9-2.8)

*Geometric mean and 95% CI in µmol/L (µg/dL).

panic blacks than for non-Hispanic whites or Mexican Americans and generally increased with age (not shown). The prevalence of elevated blood lead levels for women of childbearing age was low. Only 0.5% of women aged 12 to 49 years had blood lead levels that were 0.48 µmol/L (10 µg/dL) or greater, and this prevalence differed only slightly by race/ethnicity.

Prevalence of Blood Lead Levels 1.21 µmol/L (25 µg/dL) or Greater

In 1991, the CDC lowered the blood lead intervention level for young children from 1.21 to 0.48 µmol/L (25 to 10 µg/dL).[18] As shown in Table 3, only a small proportion of the US population (0.4%) had blood lead levels that were 1.21 µmol/L (25 µg/dL) or greater. Children aged 1 to 2 years had the highest prevalence of blood lead levels 1.21 µmol/L (25 µg/dL) or greater, and adults aged 70 years and older had the lowest. By race/ethnicity (Table 4), the highest prevalence was observed for non-Hispanic black children aged 1 to 2 years (1.4%; SE, 0.7%).

Multivariate Regression Models

Coefficients from multiple linear regression models of the log of blood lead levels, stratified by age groups, are shown in Table 6. The variation in blood lead level described by the model (R^2) was similar in the first four age groups, ranging from .27 to .31 but was lower for adults aged 50 years and older (.16). Race/ethnicity was the only variable that significantly predicted blood lead level in all of the age-specific models. The size and direction of the coefficients for the other variables were consistent across most models, but the statistical significance varied by age. In general, sex (male), urban status (central city, ≥1 million), and race/ethnicity (non-Hispanic black and Mexican American) were associated with higher blood lead levels. The PIR (high) and education (at least high school) were associated with lower blood lead levels. Age was negatively associated with blood lead level in the three models describing persons aged 1 to 19 years. In the model for adults aged 20 to 49 years, age was positively associated with blood lead level.

The sociodemographic characteristics that were significant predictors of blood lead level for children and youth dif-

Table 3.—Percentage of Population Aged 1 Year and Older at or Above Selected Blood Lead Levels by Age Category: United States, 1988 to 1991

Age, y	Blood Lead Levels, %				
	≥1.21 μmol/L (≥25 μg/dL)	≥0.97 μmol/L (≥20 μg/dL)	≥0.72 μmol/L (≥15 μg/dL)	≥0.48 μmol/L (≥10 μg/dL)	≥0.24 μmol/L (≥5 μg/dL)
1-2	0.6	1.8	3.5	11.5	40.8
3-5	0.4	0.8	2.3	7.3	28.6
6-11	0.2	0.5	1.2	4.0	16.9
12-19	0.2	0.3	0.5	1.6	8.7
20-49	0.5	0.6	0.9	3.3	21.0
50-69	0.3	1.0	1.8	7.0	34.9
≥70	0.1	0.1	0.8	6.3	38.8
All	0.4	0.6	1.1	4.5	24.1

fered only slightly by age group. Sex was not an explanatory variable among 1- to 5-year-olds but was a significant predictor of blood lead level among children and youths aged 6 to 19 years. Urban status was significantly associated with blood lead level for persons in the age groups 1 to 5 years and 12 to 19 years; among 6- to 11-year-olds, the trend was similar but not statistically significant. Income and region were both significantly associated with blood lead level for children through 11 years of age. Education of the adult reference person (less than high school) was associated with higher blood lead level in all three models. The results of these analyses indicate that for children aged 1 to 5 years, blood lead levels were highest for non-Hispanic black children from low-income families living in the central cities with population 1 million or greater. The mean blood lead level for this subgroup was 0.47 μmol/L (9.7 μg/dL) compared with 0.18 μmol/L (3.7 μg/dL) for all children aged 1 to 5 years.

Of the two adult models, the model for 20- to 49-year-olds was able to explain the greatest proportion of the variation (31%) in blood lead level, and all sociodemographic variables in this model demonstrated independent associations with the dependent variable. In contrast, the model for adults aged 50 years and older explained only 16% of the variation. Five variables (sex, race/ethnicity, PIR, urban status, and region) were significant predictors of blood lead level.

COMMENT

For the second time in the past two decades, data on blood lead levels were collected in a national survey designed to estimate the prevalence of disease and other health-related parameters in the US population. An overall geometric mean blood lead level of 0.14 μmol/L (2.8 μg/dL) indicates a substantial reduction in lead exposure since the last national survey (NHANES II, 1976 to 1980), in which the geometric mean was 0.62 μmol/L (12.8 μg/dL),[5] and represents a major public health success in primary prevention efforts to eliminate lead hazards. The decline in blood lead levels is the topic of a companion article.[19]

The findings from NHANES III phase 1 demonstrate, however, that a substantial proportion of US children younger than 6 years (8.9%) have blood lead levels now considered a health concern (≥0.48 μmol/L [≥10 μg/dL]). These levels continue to vary markedly by age, sex, race/ethnicity, urban status, income, and other sociodemographic factors. Blood lead levels were consistently higher for younger children than for older children, for older adults than for younger adults, for males than for females, for blacks than for whites, and for central-city residents than for non–central-city residents. Other correlates of higher blood lead levels included low income, low educational attainment, and residence in the Northeast region of the United States. Prevalence estimates of elevated blood lead levels from recent studies based in clinics, private, and other health care practices are consistent with the current estimates for young children.[20-24]

The variability of the estimates in this article may reflect differences in the absorption, metabolism, and excretion of lead or in the degree of environmental lead exposure. Lead is more readily absorbed by young children than by adults, but variations in other metabolic processes (eg, mobilization of lead from bone during pregnancy or during the aging process) have not been widely investigated.[25] Deficiencies in nutritional status, particularly those resulting from low iron and calcium intake, may also affect lead absorption.[26]

The primary strength of NHANES III is its ability to provide standardized estimates of blood lead levels in the US population using a high degree of both protocol standardization and laboratory QC. With the completion of the second phase of the survey, the increase in sample size will allow for a finer stratification of the population and a more comprehensive analysis of risk factors associated with lead.

A potential limitation to the design of the survey was the inability to examine the seasonal effect on blood lead levels. For logistical reasons, the mobile examination centers were located in the Northeast and Midwest in the summer months and in the South and West in the winter months. A seasonal variation in blood lead levels has been demonstrated[27] and may account for the apparent regional variability (higher blood lead levels in the Northeast) in this study. The NHANES III was also not designed to measure specific sources of lead exposure. However, sociodemographic variables can serve as indicators of the potential for lead in an individual's environment. Factors such as low income and minority status may predispose an individual to living in deteriorating, older housing or in a residential area where there is lead-contaminated urban soil and dust. Disparities in environmental lead exposure as a result of race/ethnicity, income, or geographic location have been extensively examined and are well documented.[5,26]

The public health threat posed by lead exposure may in fact be greater than the low mean blood lead levels in the general population suggest. Young children are at a greater risk for elevated blood lead levels because of their increased oral activity and ability to absorb lead coupled with the rapid development of the central nervous system in the first years of life.[26] Blood lead levels as low as 0.48 μmol/L (10 μg/dL), previously thought to be safe, have been associated with developmental delays, deficits in intellectual performance and neurobehavioral functioning,[28,29] decreased stature,[30,31] and diminished hearing acuity.[32] To address these findings, the CDC developed a multitiered approach to manage blood lead levels that are equal to or exceed the intervention level of 0.48 μmol/L (10 μg/dL).[18] Based on the results of NHANES III phase 1, approximately 1.7 million children aged 1 to 5 years in the United States are estimated to have blood lead levels exceeding this threshold. Since the publication of the current guidelines (October 1991), additional epidemiologic follow-up studies have demonstrated an inverse relationship between early exposure to low levels of lead and cognitive ability in later years.[33-35]

Of the multiple sources of exposure, lead-based paint is the principal high-dose source of lead. Exposure occurs through the direct ingestion of flaking or chalking paint or through inhalation of dust and soil contaminated with paint. Although lead-based paint was banned

Table 4.—Percentage of Children Aged 1 to 5 Years at or Above Selected Blood Lead Levels by Age Category and Race/Ethnicity: United States, 1988 to 1991

		Blood Lead Levels, %				
	Age, y	≥1.21 µmol/L (≥25 µg/dL)	≥0.97 µmol/L (≥20 µg/dL)	≥0.72 µmol/L (≥15 µg/dL)	≥0.48 µmol/L (≥10 µg/dL)	≥0.24 µmol/L (≥5 µg/dL)
All*	1-5	0.5	1.1	2.7	8.9	33.2
Non-Hispanic white	1-2	0.4	0.8	2.1	8.5	34.2
	3-5	0.4	0.4	0.7	3.7	21.3
Non-Hispanic black	1-2	1.4	5.4	10.2	21.6	63.9
	3-5	0.8	2.9	6.0	20.0	54.5
Mexican American	1-2	1.0	1.9	2.9	10.1	41.4
	3-5	0.7	0.7	1.4	6.8	34.5

*All includes race/ethnicity groups not shown separately.

Table 5.—Percentage of Children Aged 1 to 5 Years With Blood Lead Levels 0.48 µmol/L (10 µg/dL) or Greater by Race/Ethnicity, Income Level, and Urban Status: United States, 1988 to 1991

	All, %*	Non-Hispanic White, %	Non-Hispanic Black, %	Mexican American, %
Income level†				
Low	16.3	9.8	28.4	8.8
Mid	5.4	4.8	8.9	5.6
High	4.0	4.3	5.8	0.0§
Urban status‡				
Central city, ≥1 million	21.0	6.1§	36.7	17.0
Central city, <1 million	16.4	8.1	22.5	9.5
Non–central city	5.8	5.2	11.2	7.0

*All includes race/ethnicity groups not shown separately.
†Income level was defined by poverty-income ratio (PIR) categorized as low (0<PIR<1.30), mid (1.30≤PIR<3.00), and high (PIR≥3.00). Persons with missing information on income are not included in the analysis of income level.
‡Persons with missing information on urban status are not included in the analysis of urban status.
§Estimate may be unstable due to small sample size.

Table 6.—Coefficients From Linear Regression Analysis of Log_{10} Blood Lead Levels (µmol/L and µg/dL) by Age Group: United States, 1988 to 1991*

	Age, y				
Covariates	1-5	6-11	12-19	20-49	≥50
Age, y	−.04 (.01)§	−.02 (.01)§	−.01 (.01)	.01 (0)§	0 (0)
Sex					
Male	.01 (.02)	.11 (.03)§	.34 (.04)§	.34 (.02)§	.16 (.01)§
Female†	0	0	0	0	0
Race/ethnicity					
Non-Hispanic black	.14 (.03)§	.18 (.04)§	.15 (.04)§	.09 (.03)§	.12 (.01)§
Mexican American	.09 (.03)§	.12 (.05)‖	.12 (.06)	.04 (.03)	−.01 (.04)
Non-Hispanic white†	0	0	0	0	0
Poverty-income ratio	−.04 (.01)§	−.07 (.01)§	−.03 (.01)	−.02 (.01)§	−.01 (0)‖
Education‡					
Less than high school	.13 (0.2)§	.11 (.03)§	.16 (.04)§	.12 (.03)§	.02 (.02)
At least high school†	0	0	0	0	0
Urban status					
Central city, ≥1 million	.17 (.03)§	.11 (.09)	.26 (.05)§	.07 (.03)‖	.06 (.02)‖
Central city, <1 million	.09 (.04)‖	.04 (.05)	.02 (.05)	.01 (.02)	.02 (.02)
Non–central city†	0	0	0	0	0
Region					
Midwest	−.14 (.04)§	−.14 (.07)	−.16 (.12)	−.11 (.04)‖	−.11 (.03)§
South	−.22 (.04)§	−.21 (.06)§	−.17 (.10)	−.19 (.03)§	−.18 (.03)§
West	−.32 (.05)§	−.33 (.09)§	−.20 (.12)	−.14 (.05)‖	−.15 (.03)§
Northeast†	0	0	0	0	0
R^2	.27	.28	.27	.31	.16

*SEs listed in parentheses.
†Last category serves as reference category for categorical variables.
‡The education of the adult reference person was used for those aged 1 to 19 years.
§$P<.01$.
‖$P<.05$.

in 1978, deteriorating lead-based paint in residential housing continues to present a significant challenge. In addition to paint, soil and dust also act as conduits for lead deposited from gasoline emissions and industrial sources. Lead found in drinking water as a result of lead solder and pipes used in water distribution systems also presents a source of exposure for children and adults. Contaminated foods and cooking utensils as well as traditional ethnic medicines have been identified as other sources of lead exposure.[18,26]

Blood Lead Levels of Adults

Data from NHANES III phase 1 indicate that mean blood lead levels are low for young adults and higher for older adults. The distribution of elevated blood lead levels (≥0.48 µmol/L [≥10 µg/dL]) follows a similar pattern, although the proportions of levels 1.21 µmol/L (25 µg/dL) or greater were 0.5% or lower for every adult age group. The low prevalence of high blood lead levels is consistent with the fact that nonindustrial lead toxicity among adults is rare. Of adults with blood lead levels 1.21 µmol/L (25 µg/dL) or greater, it is estimated that 95% of these high levels are attributable to occupational exposure.[36] Nonetheless, occupational exposure to lead remains a concern, particularly for those who work in smelters, construction, demolition, and automobile repair.[37] The similar demographic correlates of blood lead levels across age groups suggest that sources of exposure associated with urbanization and poverty, such as deteriorated lead paint and urban dust, may be important factors for adults as well as children.

Beyond the workplace, there is an interest in understanding the potential neurotoxic effects of lead that may occur when lead is released from bone as part of the aging process.[38,39] In NHANES III phase 1, the highest geometric mean blood lead levels in adults were seen in males aged 70 years and older. If blood lead levels in the older population are more influenced by past exposure as a result of the mobilization of bone lead stores, one might expect that with recent reductions in lead exposure the levels of older persons would decrease less than the levels of younger persons. Nonetheless, the reasons for higher blood lead levels among older adults and their health significance are unclear. The low R^2 (.16) in the NHANES III phase 1 regression model for older adults underscores the need to examine risk factors that may help to explain the variation in blood lead levels

in this subpopulation. In view of studies suggesting that blood lead levels may be causally associated with higher blood pressure[40] and impaired renal function[41] in adults, further research is warranted on the relation between cumulative lead exposure and health problems associated with aging.

Blood Lead Levels of Women of Childbearing Age

The NHANES III phase 1 data indicate that mean blood lead levels for reproductive-aged females are low relative to the rest of the population. However, some data suggest an association between low blood lead levels (<0.48 μmol/L [<10 μg/dL]) measured from umbilical cord blood (which correlates well with maternal blood lead levels) and subsequent deficits in cognitive test performance and neuromotor performance in children.[28] In addition, lead from prior environmental exposure released from the bone during pregnancy may result in lead toxicity to both the mother and the fetus.[42] Unfortunately, the small number of NHANES III phase 1 women who were pregnant at the time of examination precludes a separate analysis of pregnant women and national estimates of fetal lead exposure.

CONCLUSIONS

Blood lead estimates from NHANES III phase 1 offer evidence supportive of the major achievements made in controlling lead exposure during the past decades. However, current knowledge of the health effects of low-level lead exposure underscores the benefits of past reductions at the same time as it makes clear the impact of the remaining lead exposure problem. Disproportionately, children who are minorities, poor, and living in urban areas may be at a significant risk for exposure to harmful levels of lead. Disparity in exposure to environmental hazards such as lead contributes to differences in morbidity and mortality among subgroups of the population defined by these important sociodemographic factors.[43] A concerted effort to identify the vulnerable risk groups will be vital to further reductions in lead exposure.

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